noise—is an accepted occupational hazard for the workmen. Noise dampening efforts are made, workers are encouraged to wear earplugs. But the saying is that when a millworker retires he is given not a watch but a gold-plated hearing aid. Out on our lake, motorboats are required to keep the noise of their exhausts below a certain level and underwater mufflers are mandatory.

But so far no legislator has dared suggest that mufflers be put on radios or decibel governors on stereos, even though the hearing acuity of future generations is in jeopardy. My audiologist friend suggests, half seriously, that by the year 2000 infants will be born with a hearing aid in place.

Everyone agrees we have a problem but we lack consensus on a solution. A first step might be to search for a cause of this phenomenon. A psychiatrist at the University of Chicago suggests our narcissistic younger generation turns up the volume to attract attention, the "Hey, look me over" syndrome. Another research worker maintains that the monotonous, repetitious sounds allow youths to block out the world of reality, while achieving their personal *high*. A less scholarly observation, undoubtedly made by a parent of a teenager, is that this hard-rock racket is simply an angry blast at the Establishment.

Whatever the explanation, the fact remains that these loud and pulsating sounds are an invasion of a person's privacy, as well as a cacophonous insult to his ear drums. Is there any hope for relief? The Ayatollah Khomeini's total prohibition would not work here. What we need is a forceful, grass-roots educational program.

Otolaryngologists of America, rise up in protest! Let your voices be heard. Mothers of America, make war on noise, lest universal deafness be the fate of future generations.

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Clonidine Withdrawal—Fact or Fiction?

TO THE EDITOR: The medical literature is unfortunately clouded by a maze of poorly defined terms to describe a syndrome occurring in some patients when antihypertension medication is abruptly stopped; these include discontinuation syndrome, acute posttreatment syndrome, acute withdrawal syndrome and rebound hypertension.

In a case report on clonidine withdrawal by Mate and his colleagues in the July issue (Mate TP, Swerdlin AHR, Stone RA, et al: Clonidine hydrochloride withdrawal complicating bilateral nephrectomy. West J Med 131:59-62, Jul 1979) several false and misleading statements are made concerning this syndrome. The authors attempt to incriminate clonidine withdrawal as the cause of "rebound hypertension." In the first place, the posttreatment blood pressures (250-270/100-120 mm of mercury) do not indicate any significant rebound over that of the pretreatment blood pressures (190-200/110-120 mm of mercury). Second, the authors state that "neither propranolol nor minoxidil withdrawal has been associated with a rebound phenomenon." The withdrawal syndrome is not unique to clonidine and has been reported to occur with bethanidine,[1] methyldopa[2-5] and propranolol.[6,7] Third, acute cessation of combination drugs especially a centrally acting antihypertensive in patients receiving propranolol may produce worse symptoms of blood pressure elevations than single agents alone due to uninhibited stimulation of vasoconstrictor alpha receptors during beta blockage.[8,9] The authors provide evidence that extreme levels of catecholamines appear to be the sole pressor mechanism responsible for the sustained hypertension. However, other studies have shown no excessive rise in catecholamines upon acute discontinuation of clonidine.[10,11]

It is interesting to note that abrupt cessation of clonidine (0.4 mg given orally twice a day) three days after its reinstitution in this patient did not result in another "rebound phenomenon." This is consistent with recent studies,[11,12] suggesting that doses of clonidine less than 1.2 mg per day did not result in overshoot blood pressure or symptoms of sympathetic overactivity. However, it could just as easily be interpreted that the combination of propranolol and clonidine or propranolol alone was the cause of the withdrawal syndrome in this patient. This case only adds more confusion to a poorly defined syndrome. Interchangeable use of terms such as rebound hyptertension and withdrawal syndrome should be avoided and an attempt made to distinguish between overshoot hypertension, return to pretreatment blood pressure, and sympathetic overactivity with or without hypertension. I do not believe it is justified in view of the above facts to implicate clonidine as the cause of this withdrawal syndrome. This case might just as easily

have been titled "Propranolol Withdrawal Complicating Bilateral Nephrectomy."

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Dr. Steinberg Replies

TO THE EDITOR: Dr. Houston's letter raises several important points that need clarification. Semantics are a problem in this field, but our report is not a simple case of hypertensive medication being stopped and restarted. In our patient, the cause of the hypertension, which was excessive renin from end-stage kidneys, was removed. Severe postoperative hypertension in a patient who is volume depleted by dialysis, who has no measurable renin and who still has a blood pressure of 270/120 mm of mercury, should not be confused with the more common rebound and overshoot hypertension that occurs when a patient simply stops taking his antihypertensive medication.

Although we are also familiar with the reports of severe high blood pressure when other drugs are suddenly discontinued, the cited references are not germane to our case. Dr. Houston is talking about rebound or overshoot hypertension and we described a singular circumstance in which severe and unremitting hypertension is related to catecholamines and drug withdrawal rather than a static state of sodium balance or the renin-angiotensin system. It was the uniqueness of this clinical setting of dialysis and bilateral nephrectomy that makes our remarks about clonidine and catecholamines justified. If the postoperative hypertension in this patient was not due to catecholamines or increased sympathetic discharge, what was it due to?

Finally, I doubt that our brief report could have been titled "Propranolol Withdrawal Complicating Bilateral Nephrectomy" as our patient was receiving propranolol after surgical operation and still required nitroprusside and phentolamine for acceptable blood pressure control to be achieved. This control was not accomplished until the seventh postoperative day when clonidine administration was reinstituted.

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